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CASE OF PERFORATOR INCOMPETENCE

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This is a 65-year old woman with severe recurrent venous ulcerations of the left and right legs since May 1989. She has a history of bilateral DVT 20 years ago. The patient underwent left greater saphenous vein stripping in 1978 and 1985. She has normal pedal pulses.

Level 1: There is a 10x4 cm superficial ulceration above the left medial malleolus with moderate surrounding lipodermatosclerosis. No remarkable edema.

Level 2: Duplex scanning of the left leg (May 1989) showed absence of the greater saphenous vein. The common femoral and profunda femoris veins were patent and competent. There was partial recanalization of the superficial femoral and popliteal veins. The posterior tibial vein was also recanalized. No perforating veins were identified. Duplex scan Sep 1997: left superficial femoral and popliteal veins patent but partially compressible, posterior tibial vein incompetent, lesser saphenous vein incompetent, two incompetent perforators medial calf. APG Sep 1997: OF 16%, VV 68 ml, VFI 5.1 ml/sec, EF 65%, RVF 44.1%.

Level 3: Descending venography reveals valvular incompetence in the common femoral and proximal superficial femoral veins; contrast flows retrograde to the level of mid-superficial femoral vein. Lymphoscintigraphy: no abnormality left lower extremity.

Duplex Mar 1998: no change from Sep 1997. Three incompetent perforating veins medial calf.

CEAP Classification: C6s; Es; As,p,d; Po

Treatment?
(see figures 1-2, on p. 264)

DISCUSSION

DR. O'DONNELL: I do have a little problem with the use of the eponym "Cockett" for these operations. Cockett, as you know, originally described an extrafascial approach to perforators and reserved the subfascial approach for severe dense lipodermatosclerosis with ulcer. Actually, Dodd, Cockett's co-author of their classic text, abandoned the extrafascial approach very early on his experience because of wound complications. In addition he moved the incision postero-laterally. So what you call Cockett is not what Cockett himself described.

DR. O'DONNELL: This is a very interesting case, certainly not one of straightforward perforator incompetence in that there seems to be an element of deep venous obstruction. Our panel had very interesting responses. I question you, gentlemen and ladies, can you provide any evidence that doing something to the perforating veins is going to make this patient better? I would submit that no one in the audience can show in a case like this that the hemodynamics improve. Indeed, most of the data in the literature shows no hemodynamic improvement in patients with post-thrombotic syndrome following interruption of the perforators. Going back to some of the early studies by our Scandinavian colleagues - occlusion of a perforating vein and measurements with electric magnetic flow meters and venous pressures showed no improvement in hemodynamics. And our own work confirms the same. Therefore, I find it very interesting in this case that we're going to treat the perforators alone, but I don't know to what end. Let me open it up to the panel after these "prejudicial" statements. Peter, from your North American SEPS Registry study you have a one in two chance at least with a short-term follow-up of having a satisfactory result i.e., -no ulcer recurrence, in this case if you interrupt the perforators; right?

DR. GLOVICZKI: Well, this is a difficult case, and I seldom perform perforator ligation in a patient with deep vein obstruction or with an element of deep vein obstruction. In this patient obstruction has been confirmed by APG studies. Unfortunately, we do not have an adequate evaluation of this patient. Ultimately, I think that I am going to suggest SEPS, but I would probably make another attempt of an ascending venography. I think an ascending venography in this patient would be quite critical.

DR. O'DONNELL: Why don't you show the ascending phlebogram that you did do, Paul - after the procedure?

DR. GLOVICZKI: You should have done the ascending venogram before the procedure.

DR. DEPALMA: One question that I missed completely is the status of the lesser saphenous.

DR. CORDTS: The lesser saphenous vein was incompetent

DR. DEPALMA: It was incompetent. Okay. That's important because the lesser saphenous gives an Achillean perforator as it crosses the tendon initially to Cockett 1. That is what Dr. Enrici's arcade shows as he dissects. I think that it's very important to deal with that inflow problem as well as interrupting perforators from above.

DR. O'DONNELL: Ralph, how do you deal with the incompetent lesser saphenous? Do you strip it out? Do you ligate it? What do you do?

DR. DEPALMA: Well, I think all of the action is down at the lower end, and I would divide it. I would ligate it and then just put the small skin incision out of the area of involvement and then come down directly on the Cockett 1, ligate that, remove the Achillean communication. Then I elevate the skin around it and then dress the dissected area firmly and keep the limb elevated.

DR. GLOVICZKI: I like invagination stripping of the lesser saphenous vein. I think it is nontraumatic and it preserves the sural nerve. These are frequently perforator veins connecting the lesser saphenous vein to the deep veins, so stripping is a better operation than ligation only.

DR. NEGLEN: I would like to turn this case around. If I understood it right, you had axial reflux in the superficial femoral vein that was patent and partially recanalized. So if we forget the perforators and then look at axial reflux in the deep system in a limb with stripping of the saphenous vein already performed, reflux flow

on the APG as high as 5.1 ml/s is sort of high for perforator incompetence, even with the popliteal vein incompetence. That's the first point. The other point is that I would also be very interested in looking at the iliac vein and agree with Dr. DePalma, since the outflow fraction is only 16 percent. Although I don't trust the measurement of outflow fraction per se, 16 percent is very low and probably positive for obstruction. So I would say, isn't this a case of axial deep vein reflux with outflow obstruction, although I don't know where the obstruction is?

DR. CORDTS: The obstructive changes were in the superficial femoral vein and popliteal veins. The iliac veins looked normal by venography.

DR. NEGLEN: Completely normal by venography? Then I would suggest you perform a valvuloplasty of some sort of the superficial femoral vein, which would probably lead to a better result than a SEPS.

DR. O'DONNELL: What would the panel say to that? That would be my conclusion, but I'm glad you stated it. Treating the perforators alone at least hemodynamically does nothing.

DR. DALSING: In this case, I'm more concerned that the obstruction is important. Even if you couldn't obtain an ascending venogram, I think something like a magnetic resonance venogram, may be possible. This would allow you to look at the anatomy in some way. I am worried about the obstruction. I'm not so sure that putting a valve in this system to prevent reflux is going to take care of an APG of 16 percent. I don't think I've even seen a false positive APG to that degree. Yes, I have seen false negatives, but not a false positive. These are the things that I'm concerned about when considering this case.

DR. KISTNER: I thought you said that the reflux only went to the lower thigh and not down into the popliteal. Am I wrong?

DR. CORDTS: Reflux to the lower thigh on the descending venogram but then on duplex scan subsequent to that it went down more distal than that into the popliteal vein and the lesser saphenous vein.

DR. KISTNER: And how did it get there?

DR. CORDTS: It got there through the superficial femoral veins. Those studies were done at different times.

DR. DALSING: Was that an obstruction or a valve present there?

DR. CORDTS: An obstruction in the superficial femoral vein.

DR. KISTNER: Did you consider this a problem of reflux or a problem of obstruction? Could you separate those, or was it both?

DR. CORDTS: Initially obstruction and later reflux, later both.

DR. KISTNER: I don't see repairing the reflux unless you could demonstrate that it has significant volume.

DR. NEGLEN: Bob, is it occlusion and is it recanalization and to what degree do we have the lumen? I can understand the hesitancy of doing a valvuloplasty above an occlusion, but we'd really like to see those venograms.

DR. KISTNER: If there's significant reflux it should be eliminated. It could either be by putting a valve or by ligating the SFV.

DR. DEPALMA: If I can make a comment here, 30 cc's of dye is worth three opinions. I'm used to looking at arteriograms and venograms. I'm not so smart in guessing at ultrasound or physical examination. We've had this discussion about the use of duplex scans to do operations, and in the recalcitrant group of patients the venous system is pretty complicated. Here's Sherman's depiction of the saphenous and branches published in the 1950's. We tend to forget how complicated the superficial system is and how many branches there are. If you can figure that out on duplex, especially when the skin is thick and a big ulcer exists, I don't know how do it. I think this is the usual end result of surgery rather than conservative therapy. I have had a problem correlating our duplex scans, which are wrong about 30 percent of the time, in making operative decisions. This case is a perfect example of that. In this case the surgeon has left the saphenous behind along with missed perforators. I repeat again that 30 cc's of dye is always worth three opinions.

DR. O'DONNELL: I would agree that when you get a complex venous case that duplex alone is insufficient. It's our routine to get ascending and descending phlebograms, particularly in a patient with an ulcer.

DR. KISTNER: Certainly I'd have ascending and descending venography to map out everything in the leg. It looks like there's a good chance that the superficial femoral vein is contributing reflux, and I'd eliminate that, probably repair it, and if I couldn't repair it I'd probably ligate it. I'm not clear what's going on in the profunda femoral vein, and I think you need a descending venogram to find out what the flow patterns are. So often you find a different flow pattern with descending venography than you do with ascending venography, and if you add the two together and throw in the duplex, you get a picture. So I would analyze this case more completely, and fix what I could fix. I guess that reflux is the key more than obstruction.

DR. RAJU: I would totally agree with what Bob said. I mean, it's clear that the superficial femoral vein is ten times as large as any perforator we saw, and I think it would be a mistake to focus on the perforators. Dr. Gloviczki has been presenting data in the last few days in this very meeting saying that one-year recurrence is about 35 or 30 percent, in post-thrombotic syndrome is it not, Peter?

DR. GLOVICZKI: The two-year data was 46 percent, but that had a large percent of standard error because we didn't follow too many patients up to 2 years. Post-thrombotic patients do the worst.

DR. RAJU : So you have already done SEPS.

DR. GLOVICZKI: You have not. You did a sham operation.

DR. RAJU: It has already failed, and you have shown persistence, or new formation, of perforators. You saw some other unnamed saphenous branch taking in a perforator up in the thigh. I think it has been shown in the venous system that you cannot disconnect and isolate the superficial or some part of the venous system from the other permanently. This has been amply shown in the portal circulation. Warren operation is based on that. It works for four or five years. After that you get reconnection. That should be the time for SEPS. I think all the disconnecting operations are going to be temporary. Preliminary SEPS data shows the recurrence to be high in post-thrombotic cases. The superficial femoral vein is large in this case. There is massive reflux, and I do not understand the hesitancy to go and fix that reflux.

DR. O'DONNELL: Peter, would you comment? You said a sham operation. Is that because the lamina profunda was not incised in the posterior compartment?

DR. GLOVICZKI: I was joking. It was not a sham operation. It was just not a complete operation and that was obvious. In such a short time those large perforators don't just show up. I mean, obviously if the deep posterior compartment was not entered,

several important perforators were missed. So it was an incomplete operation. This patient has severe post-thrombotic reflux and obstruction. I would start out with a redo SEPS, but I don't debate that this patient will ultimately really benefit from a good operation to correct the deep reflux. The debate could be whether it's femoral ligation as Bob suggests, or something else. The question is if the valve is not reparable would you do an axillary vein valve transplant or would you, in a patient like this, put in a cryopreserved vein valve that has a 60 percent thrombosis at six months.

DR. PERRIN: I think the ulcers recurred after SEPS. I would propose ultra-sound guided technique for the perforators. That's the first thing. If that does not work, I would probably redo SEPS.

DR. O'DONNELL: You wouldn't treat the deep system?

DR. PERRIN: No.

DR. DALSING: I guess I would approach the deep system and try to repair it, like Bob says. The actual six-month patency rate for cryopreserved vein valves is probably in the 60 percent range, not the 40 percent patency rate suggested by Peter.

DR. KISTNER: If that profunda system is patent and competent, it's a different ballgame than if it's diseased or absent. So you've got to find that out because that tells you whether it's worthwhile to fix the superficial femoral system, I think.

DR. NEGLEN: I think this is very important what Bob said. We still don't know enough about the axial reflux. Seeing the films I got a feeling there is a sort of profunda transformation and maybe it wasn't the main superficial femoral vein we saw. The second point I want to return to is the low outflow fraction and the suspicion of outflow obstruction. I think this patient needs a trans femoral venogram which clearly shows the iliac segment. This segment can't be assessed in this film although it appears normal. I agree with Dr. Dalsing that 16 percent outflow fraction is very low and it's rare to have false positive findings of that magnitude. There is something cooking up there.

DR. DALSING: When you did your descending venogram did they look at the iliac when they went down?

DR. CORDTS: Yes.

DR. DALSING: And was it normal then?

DR. CORDTS: Yes.

DR. O'DONNELL: I personally would get a complete venogram and do an arm-foot vein pressure study to determine the elements of obstruction.

SURGICAL MANAGEMENT

DR. CORDTS: Let me show you what we did first. In March of '98 we did a SEPS. We did a SEPS using the standard techniques that have been described. We identified three incompetent perforating veins by duplex scan and marked them pre-operatively. Then we identified those, clipped them and divided them. We did not open the deep posterior space. Then we exposed the lesser saphenous vein at the saphenopopliteal junction and tried to but couldn't pass the PIN stripper. So we treated the lesser saphenous vein by ligation and division since we could not strip the lesser saphenous vein. Over the next few months she showed improvement but three small venous ulcers remained. In August 1999, a year and a half later, the ulcers had never healed. We did a duplex scan which showed the common femoral vein and popliteal vein were incompetent. The tibial veins

Figure 1.– Ascending venogram left calf, oblique view. Venogram demonstrates two residual incompetent perforating veins medial calf (white arrows). Clips from prior SEPS procedures are noted in more distal calf.

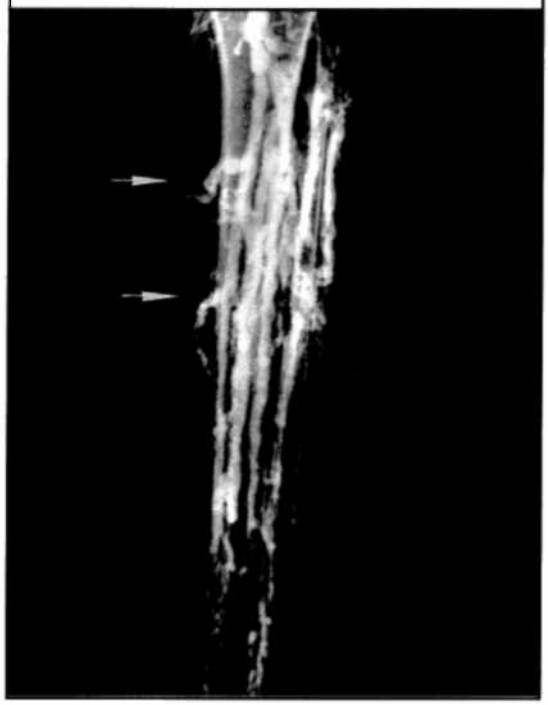

Figure 2.– Ascending venogram left leg, lateral view. Venogram demonstrates multiple incompetent thigh perforating veins (white arrows) filling superficial varicosities. Post-thrombotic changes of distal superficial femoral vein are noted.

appeared competent, and we identified three incompetent perforating veins. This is a year and a half after SEPS. We then did an ascending venogram. We were able to identify three incompetent perforators (Fig. 1). You can see the clips from the SEPS, and you can see that one of those perforators communicates with an incompetent segment of the greater saphenous vein below the knee. So that's not good. There were recanalization changes of the superficial femoral vein (Fig. 2), and an incompetent perforator in the thigh are seen. Then as you come up in the groin here we identify some portion of the profunda, and the remainder of the iliac veins appear to be normal. So we identified three incompetent perforators in the calf, at least one incompetent perforator in the thigh, and recanalization changes of the superficial femoral vein. We haven't done anything further at this point.

III. PRIMARY VENOUS DISEASE: DEEP AND SUPERFICIAL REFLUX

TREATMENT OF PRIMARY VENOUS INSUFFICIENCY

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Recognition that a chronic venous problem is due solely to primary venous insufficiency (PVI) defines that the entire problem is due to reflux in the veins, and that obstruction is absent. PVI cases can present with exactly the same clinical appearance as post-thrombotic cases, but the treatment implications are quite different because the venous system is entirely patent and the luminal surfaces of the veins are normal. Successful treatment is possible by surgical means in all segments of the lower extremity veins afflicted with PVI, including superficial (saphenous), perforator, and deep veins, and the results in all of these divisions of the venous tree are highly favorable.

PVI has distinctive histologic findings that have been published in the past but are not frequently appreciated. The findings in the endothelial layer consist of hyperplasia, which produces the white streaks often seen on the luminal surface of the opened vein. The real pathology is in the subendothelial and medial layers where the most striking change is an increase of the collagen which appears to become aggressive by wrapping around muscle bundles and actually fragmenting the syncitial continuity of the muscles in PVI. This is accompanied by fragmentation of elastic layers and areas of either hyper- or hypoplasia of the muscular layers. This process logically results in dilation of the venous wall, and dilation of the wall leads to valve incompetence.

The striking difference between primary and secondary disease is that the signs of prior acute thrombosis and inflammation or hemorrhage seen in post-thrombotic disease are absent in primary disease. These signs are hemosiderin deposition, neovascularization in the old thrombus and in the vein wall, and leukocyte infiltration of the wall.

The gross changes of PVI are strikingly different than those found in post-thrombotic disease (PTD). In PVI, the lumen is smooth and the wall is pliant and of relatively normal thickness. Valve sites are fewer in the saphenous vein of PVI than in the normal state, probably due to atrophy and ultimate disappearance. All stages of atrophy of valve cusps can be seen in these veins. In the deep veins, the valve cusps are normal in appearance, but are stretched and elongated. These findings are strikingly different than in post-thrombotic veins where the luminal surface is irregular, contains synechiae and random webs, and sometimes endoluminal masses are present. The valves are disfigured, scarred, and often entirely destroyed. The wall is thickened, non-pliant, and there is usually a peri-phlebitis with adhesions to surrounding tissues.

Given these differences, it is not surprising that there are excellent opportunities for surgical repair in PVI and little reason for limiting treatment to external support and change of life-style in the otherwise healthy person. With care in diagnosis, and adherence to the CEAP requirements for definition of the etiologic basis for the clinical problem between primary, secondary, and congenital causes, and the pathogenetic mechanisms of reflux and obstruction segment by segment, opportunities for correction of the abnormal physiology abound in primary disease.

Treatment of superficial primary disease in the saphenous system, and of the perforator veins, is widely practiced and is all that is needed in 30-50% of the cases of ulceration, and in the vast majority of non-ulcer cases. The deep system is implicated in 60%+ of primary ulcer cases, and requires surgical repair in a so-far unknown percentage of these to provide long-term relief of the Class 4, 5, and 6 problems. The long-term success of valve repair, coupled with control of saphenous and perforator incompetence in PVI cases of classes 4, 5, and 6 up to 4 years and beyond, is well-demonstrated in the literature to fall in the range of 65-80% in every published series of significant size.

Given the present ability to diagnose primary venous disease accurately and by non-invasive affordable tests, the appropriate management of all primary disease should be by surgical correction in the active patient. This includes saphenous, perforator, and deep vein correction. The major question is to define which patients do, and which patients do not, require correction in the deep veins to provide a long-lasting favorable result. The answer to this will require comparative prospective treatment groups.

(Scientific Articles continue on next page)